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Award Number: W81XWH-09-1-0284

TITLE: Minority Undergraduate Research in Prostate Cancer: Bridging Opportunities for Postbaccalaureate Education

PRINCIPAL INVESTIGATOR: Robert Sikes, Ph.D.

CONTRACTING ORGANIZATION: University of Delaware  
Newark, DE 19716

REPORT DATE: April 2011

TYPE OF REPORT: Annual Summary

PREPARED FOR: U.S. Army Medical Research and Materiel Command  
Fort Detrick, Maryland 21702-5012

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REPORT DOCUMENTATION PAGE				Form Approved OMB No. 0704-0188	
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1. REPORT DATE (DD-MM-YYYY) 01-04-2011		2. REPORT TYPE Annual Summary		3. DATES COVERED (From - To) 1 Apr 2010 - 31 Mar 2011	
4. TITLE AND SUBTITLE Minority Undergraduate Research in Prostate Cancer: Bridging Opportunities for Postbaccalaureate Education				5a. CONTRACT NUMBER	
				5b. GRANT NUMBER W81XWH-09-1-0284	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S) Robert Sikes, Ph.D.  E-Mail: rasikes@udel.edu				5d. PROJECT NUMBER	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) University of Delaware Newark, DE 19716				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES) U.S. Army Medical Research and Materiel Command Fort Detrick, Maryland 21702-5012				10. SPONSOR/MONITOR'S ACRONYM(S)	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION / AVAILABILITY STATEMENT Approved for Public Release; Distribution Unlimited					
13. SUPPLEMENTARY NOTES					
14. ABSTRACT Four students, 3 from Delaware State University and 1 from Lincoln University, were recruited to participate in prostate cancer research in laboratories at the University of Delaware. In compliance with the aims of our grant the students each received intensive research training over the 10-week summer program. All students were required to participate in enrichment activities that spanned the scope of intellectual property, careers in medicine and science, as well as good research practice. Also in compliance with our aims, this grant sponsored three Health Disparity round table discussions that covered a range of issues in minority health. These discussions included topics of access, economic status, racial profiling, provider perceptions/misconceptions and race-based medicine. All discussions were based on primary literature as a lead in to the topic followed by group discussions. All students presented posters in a research symposium with over 500 participants from UD, Wesley, UMBC and other regional schools. Selected students participated in regional competitions.					
15. SUBJECT TERMS Undergraduate students, minority, abstracts, awards					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT  UU	18. NUMBER OF PAGES  20	19a. NAME OF RESPONSIBLE PERSON USAMRMC
a. REPORT U	b. ABSTRACT U	c. THIS PAGE U			19b. TELEPHONE NUMBER (include area code)

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**Introduction:**

Due to the extremely low levels of minority faculty and graduate students in the sciences, the DoD Majority Institution (MI) /Historically Black College and University (HBCU) program was intended to foster and promote the interest of minority students in basic science and research by partnering one or more HBCU with a sponsoring MI. In Delaware, this has been accomplished by coordinating student recruitment from Delaware State University and Lincoln University to perform funded summer research in prostate cancer laboratories at the University of Delaware. Our Aims were to 1) offer a 10-week summer research program to five qualified minority students, 2) Offer a summer enrichment program to these students and 3) offer activities and extended research at the participating HBCUs during the following academic year.

**Body:**

In compliance with Aim 1, and upon the recommendation of the faculty campus coordinators at Delaware State University (Dr. Cindy van Golen) and Lincoln University (Susan Safford), 4 students from DSU and 1 student from Lincoln University were chosen for admission into the University of Delaware's training program in Prostate Cancer after being interviewed by prospective faculty mentors at UD. Our HBCU facilitators have been having an increasingly difficult time with this task due to high demand, or competition from other funded summer programs recruiting minority students nationally.

<b>Student</b>	<b>School</b>	<b>UD Mentor</b>	<b>Project</b>
Jhoneil Cooper	DSU	John Koh	Soft Agar Colony Formation Assays with LNCaP and CWR22 in the presence of PLM1, PAN52 and PLM6 compared to Bicalutamide
Jennifer Gray	DSU	Randy Duncan	The Role of the microRNA endonuclease Dicer in Sea Urchin Embryology
Odinaka Anyanwu	LU	Kirk Czymmek	
Navpreet Tung	DSU	Robert Sikes	HS-5 Bone Marrow Stromal Cell Conditioned Media may Promote Cell Cycle-Dependant Cell Death in Prostate Cancer through TGF- $\beta$ Signaling
Ruth Joanis	DSU	Ken Van Golen	The Connection Between IGF-1 and the Activation of Different Rho GTPases and their Connection to Cell Invasion and Metastasis of Prostate Cancer

In compliance with Aim 2, students attended weekly seminars related to research <http://www.udel.edu/chem/white/HHMI3/Summer10/S10enrichment.html>. In addition our students attended discussion sessions on the topic of *Healthcare Disparities*. Prior to each session students were assigned to read both popular and scientific literature regarding the socio-economic or medical causes of healthcare bias. UD faculty from the Departments of Biological Sciences, Chemistry and Biochemistry moderated the discussions.

In compliance with Aim 3, Dr. van Golen and Dr. Usher presented seminars at DSU and Lincoln respectively. One student, Navpreet Tung, continued to perform bench science at UD through the next three semesters.

**Key Research Accomplishments:**

The students in this program were expected to make significant progress in research over a 10 week period. Students instructing them in laboratory procedures that included basic liquid handling, safety, and use of technology and equipment required. Despite this, the amount of publishable data that each student collected during this short time is amazing. Additionally, students were instructed to journal their research experience to enhance their level of comfort of communicating what skills and techniques they learned as well as understanding the research project. At the end of the summer program, each student presented the results of their research at the University's undergraduate research symposium, which required the students to produce a written abstract and poster for presentation. The symposium was modeled after the Experimental Biology meeting, where posters and talks occurred simultaneously and where there was a plenary lecture by a Howard Hughes Medical Institute investigator <http://www.udel.edu/chem/white/HHMI3/Summer10/S10enrichment.html>.

Navpreet Tung received a first place in his division at the UMBC (University of Maryland Baltimore County) undergraduate Research symposium.

**Reportable Outcomes:**

Five posters and 1 regional competition award

**Conclusions:**

Our students frequently state that their summer experience has made them evaluate research as a career option. In many cases this has resulted in graduate school applications instead of vocational programs in Nursing or other health related field. The students leave excited and we have had many students who apply for a second year. This fact alone suggests that we have a viable, rewarding program that is not redundant or repetitious from year to year. We are producing students of quality from HBCUs who can compete regionally and win prizes in poster competitions based on their results.

**References:**

None

**Appendices:**

Enrichment program schedule (Summer 2010)

Abstracts-Used for multiple meetings as described above (5)

Posters with each student presenting at year-end symposium (5)

UMBC poster competition news article

# SUMMER 2010 UNDERGRADUATE RESEARCH ENRICHMENT PROGRAM

## Tentative

Wednesdays 4 to 5 PM 205 Brown Lab



**HHMI**  
HOWARD HUGHES MEDICAL INSTITUTE

Special Sessions Thursdays June 10  
4-5:30 PM

June 24 & July 28 NOON-1:30 PM

HHMI Scholars, Peter White  
Fellows, Beckman Scholars,  
and others including Science  
and Engineering Scholars

DATE	PROGRAM
On-line On your own	<i>What do you need to know about Safety in the Research Laboratory?</i> <b>Please note:</b> You need to have completed safety instructions in your research laboratory and/or <u>on-line</u> before you start work in a laboratory. If you have questions, contact <u>Occupational Health and Safety</u> .
Thurs June 10 4:00-5:30 Gore Hall	<i>Undergraduate Research Ethics Conference</i> <u>Dr. Thomas Powers</u> , Department of Philosophy and co-director of the <u>Science, Ethics, and Public Policy program</u> administered by the Delaware Biotechnology Institute, and graduate students
June 16	<i>What are you doing here this summer? Introduction to Research and issues you may encounter.</i> <u>Dr. Harold White</u> (Dept. Chemistry & Biochemistry, Director UD's HHMI Undergraduate Science Education Program). <u>Dr. David Usher</u> (Dept. of Biological Sciences, Assoc. Dir. UD's HHMI Undergraduate Science Education Program)

<b>June 23</b>	<p><i><b>Student Voices. Students who have done undergraduate research for more than a year describe their experiences.</b></i></p> <p><u><a href="#">Megan Kissig</a></u>, <u><a href="#">Nick Marze</a></u>, <u><a href="#">Michael Napolitano</a></u>, <u><a href="#">Wuroh Timbo</a></u>, and <u><a href="#">Justin DiAngelo</a></u> BS Biology '02, PhD UPenn '10 (F 2010 begins Asst. Prof. Cell Biology at Hofstra University.)</p>
<b>Thurs June 24 (optional)</b>	<p><i><b>Dealing with America's Health Disparities Problem - Part I Socioeconomic and Cultural Factors</b></i></p> <p><u><a href="#">Drs. David Usher</a></u>, <u><a href="#">Robert Sikes</a></u> (Dept. of Biological Sciences), <u><a href="#">Jacqueline Aldridge</a></u> (NUCLEUS Program), and <u><a href="#">Cynthia van Golen</a></u> (Delaware State University), and <u><a href="#">Susan Safford</a></u> (Lincoln University)</p> <p><i><b>Special optional session in 243 Wolf Hall from 12-1:30 PM. Food Provided.</b></i></p> <p>Readings: <u><a href="#">Disparities and Discrimination in Health Care-an Introduction</a></u> <u><a href="#">Health Care Disparities Reading List Abstracts.</a></u></p>
<b>June 30</b>	<p><i><b>Don't stop now-Other University opportunities?</b></i></p> <p><u><a href="#">Susan Serra</a></u>, Service Learning Coordinator and <u><a href="#">Katharine Kerrane</a></u>, Senior Associate Director, Honors Program, and undergraduate panelists discussing National and International Scholarship Opportunities, Semester Abroad, Service Learning, and related opportunities. (<u><a href="#">Goldwater</a></u>, <u><a href="#">Marshall</a></u>, <u><a href="#">Mitchell</a></u>, <u><a href="#">Rhodes</a></u>, and <u><a href="#">Truman</a></u> Scholarships, <u><a href="#">Fulbright Fellowships</a></u>)</p>
<b>July 7</b>	<p><i><b>How are things going? Mid-Session Perspectives.</b></i></p> <p><u><a href="#">Dr. David Usher</a></u> (Dept. of Biological Sciences, Assoc. Dir. UD's HHMI Undergraduate Science Education Program), <u><a href="#">Dr. Harold White</a></u> (Dept. Chemistry &amp; Biochemistry, Director UD's HHMI Undergraduate Science Education Program).</p>
<b>July 14</b>	<p><i><b>How do I get into Graduate School? (Must attend this session and/or the July 21 session)</b></i></p> <p><u><a href="#">Dr. David Usher</a></u>, (Dept. of Biological Sciences), <u><a href="#">Dr. Melinda Duncan</a></u>, (Dept. of Biological Sciences), <u><a href="#">Dr. Brian Bahnson</a></u>, (Dept. of Chemistry and Biochemistry) <u><a href="#">Dr. John Pelesko</a></u>, Dept. of Mathematical Sciences)</p>
	<p><i><b>Dealing with America's Health Disparities Problem- Part II</b></i></p>

<p><b>July 20</b> <b>Tuesday</b> <b>lunch</b> <b>(Optional)</b></p>	<p><b><i>Race-based Medicine</i></b>  <u><a href="#">Drs. David Usher</a></u> and <u><a href="#">Robert Sikes</a></u> (Dept. of Biological Sciences),  <u><a href="#">Jacqueline Aldridge</a></u> (<u><a href="#">NUCLEUS Program</a></u>), <b>Dr. Cynthia van Golen</b>  (Delaware State University), <u><a href="#">Dr. Susan Safford</a></u> (Lincoln University)  <b>Special optional session in 243 Wolf Hall from 12-1:30 PM</b>  Reading: <u><a href="#">Should Racial Profiling have a Role in Cancer Prognosis?</a></u></p>
<p><b>July 21</b></p>	<p><b><i>Managing a career in science. What is it like to be a scientist in academia or industry?</i></b>  <b><i>Career biographies from academic and industrial scientists.</i></b>  <u><a href="#">Erica Selva</a></u> and <u><a href="#">Kenneth van Golen</a></u>, Department of Biological Sciences,  <u><a href="#">Charles Riordan</a></u>, Department of Chemistry and Biochemistry, and  <b>Easley Wallace, Jr.</b>, Principal Investigator, DuPont.</p>
<p><b>July 28</b></p>	<p><b><i>How to communicate your Results - Conferences (<u><a href="#">Talks</a></u> and <u><a href="#">Posters</a></u>)</i></b>  <u><a href="#">Megan Kissig</a></u>, BS Biology, and <u><a href="#">Michael Napolitano</a></u>, BS Biochemistry  <b><u><a href="#">Judging Rubrics</a></u> for the ASBMB Undergraduate Poster Competition 2007</b>  A good site for <u><a href="#">instructions on poster preparation</a></u>. Another <u><a href="#">good site</a></u>.</p>
<p><b>August 4</b></p>	<p><b><i>How do I get into Medical or other professional Schools? (Must attend this session and/or the July 14 session)</i></b>  <u><a href="#">Dr. David Usher</a></u> (Dept. of Biological Sciences), <u><a href="#">Aivi Nguyen</a></u>, BS Biology '09 (Jefferson Medical School), <u><a href="#">Obi Mmagu</a></u>, BS Biology '09 PCOM,  <u><a href="#">Christine Arenson, MD</a></u> BS Chemistry '86, Director, Division of Geriatric Medicine, Jefferson Medical School.</p>
	<p><b><u><a href="#">Undergraduate Research and Service Celebratory Symposium</a></u></b></p>



**Aug 11**



**Catherine Drennan**

Professor of Chemistry

Massachusetts Institute of Technology

HHMI Professor

HHMI Investigator

Teaching General Chemistry with a Biological Emphasis

**Plenary Lecture:**

**Snapshots of Proteins in Action**

11:15 A.M.

Clayton Hall

9:00 - 11:00 AM

(Participants and their Poster Assignments to be posted in August)

**Student Talks and Posters Presentations**

**Clayton Hall**

**(Last year's program)**

[HHMI Undergraduate Research](#), [University of Delaware Undergraduate Research Program](#), [HHMI Home Page](#)

Program organized by David Usher [e-mail: [dusher at udel.edu](mailto:dusher@udel.edu)], [Department of Biological Sciences](#)

Page last updated: 22 July 2010 by [Hal White](#) [e-mail: [halwhite at udel.edu](mailto:halwhite@udel.edu)], [Department of Chemistry and Biochemistry](#)

## **Jhoneil Cooper and John Koh**

### **Soft Agar Formation Assays with LnCap and CRW22 cells in the presence of PLM1, PAN52, and PLM6 compared to Bicalutamide and Flutamide**

#### **Introduction**

Prostate Cancer is the second leading cause of cancer death in men. As prostate tissue is dependent on androgens for growth, anti androgens used alone or in conjunction with inhibitors of testosterone biosynthesis have been used in the treatment of Prostate Cancer however, often cancer cells escape such androgen blockade therapies. Antiandrogen failure can be caused by incomplete AR inactivation by antiandrogens caused by androgen receptor mutations, androgen receptor overexpression and or cytokine signaling crosstalk are associated with antiandrogen failure. Antiandrogen failure often leads to a clinical phenomenon known as anti-androgen withdrawal syndrome wherein anti-androgen resistant patients show symptomatic improvements after cessation of anti-androgen treatment. To compare compounds with each other, as PLM1 is bad in clonogenic assays where as PLM6 is good. Also compare the cells that were plated and to compare the other reactions to that of Bicalutamide. PLM6 and PAN52 will react better with cells to form more resistant colonies in the soft agar colony assay. We will observe a difference in the incidence of resistant colonies between antiandrogen naïve CWR22 cells compared to flutamide resistant LNCaP cells if our second generation antiandrogens are acting through a non-antiandrogen specific resistance pathway. This experiment entails the assessment of colony formation of LnCap and CRW22 cells within the presence of the above mentioned drugs including a control. Cells will be plated in 6 well plates and a soft agar assay will be carried out which can be described as trapping the cells within a gel while testing them with specific compounds. The cells will then be allowed to grow for a two week period during which pictures of the cells will be taken to determine their reaction to the experiment and to also gather the results of the experiment.

Jennifer Gray, Mary Boggs, and Randall Duncan

Bone is highly innervated by sensory and sympathetic neurons which most believe are primarily implicated in controlling vascular activity. While the claim that these nerve fibers are purposefully located to regulate blood flow, studies have revealed the possibility of these neurons involvement in regulating bone structure (source-MB). This theory is supported by the discovery that the removal of sympathetic nerve fibers from bone leads to disregulation of the bone remodeling process. This phenomenon is indicative that neurons communicate with bone cells directly. However, the manner by which this communication system occurs is unknown. Thus, this study's aim is to investigate the effects neurotransmitters may have on osteocyte activity by the use of calcium imaging techniques. MLO\_Y4 cells were treated independently with 100 $\mu$ L of GABA, Epinephrine, and glutamate agonist NMDA and AMPA.

## The Role of the microRNA Endonuclease Dicer in Sea Urchin Embryology

Odinaka C. Anyanwu<sup>1</sup>, Deborah H. Powell<sup>2</sup>, Jia L. Song<sup>3</sup> and Kirk J. Czymmek<sup>2,3</sup>

<sup>1</sup> Lincoln University of Pennsylvania, Oxford, PA <sup>2</sup>Delaware Biotechnology Institute, Bio-imaging Center, University of Delaware, Newark, DE <sup>3</sup>Department of Biological Sciences, University of Delaware, Newark, DE

Dicer is an RNaseIII type endonuclease and is responsible for the regulation of microRNA. MicroRNAs are non-coding RNAs about 22 nucleotides in length. Specifically in adenocarcinoma, a form of prostate cancer [1], several are found to be up-regulated and directly proportional to the severity of the cancer. Conversely, it has also been shown that down regulation of Dicer expression in mice showed an enhanced tumorigenesis phenotype [2]. Considering this strong link of Dicer to cancer as well as microRNAs implicated in other cancers such as lymphomas, breast cancer, lung cancer and more, [3] a thorough evaluation of how it is regulated and how alterations in its expression affect cells is needed. For this research, sea urchins were used as a model system to evaluate the role of Dicer during embryonic development. Due to its optical transparency, the sea urchin embryo is very well-suited for microscopy experiments allowing delineation of cell types expressing Dicer in the entire embryo. Furthermore, its full genome recently has been sequenced and found to have significant homology to many important proteins in vertebrate biology. The aims of this project were to develop a method for localization of Dicer and 3D rendering of intact embryos with the subsequent identification of specific cell-types. Preliminary data suggested that Dicer may have a specific asymmetrical localization pattern during early gastrula development, while later stages lacked asymmetrical distribution.

Navpreet Tung, Fayth L. Miles, Robert A. Sikes

Prostate cancer (PCa) is the second leading cause of cancer death in North American men. Aggressive PCa metastasizes to bone and is characterized by high levels of Transforming Growth Factor- $\beta$  (TGF- $\beta$ ) in serum. We have shown that TGF- $\beta$  reduces the growth of both bone-adapted and bone naïve PCa cell lines, although the mechanism has not been elucidated. Additionally, we have shown that the human bone marrow stromal cell line, HS-5, secretes a factor toxic to PCa cells, leading to increased cell death. This hostile bone stroma: PCa interaction is mediated through a TGF- $\beta$  family member, as it is abrogated by SB-431542, an inhibitor of TGF- $\beta$  type I receptors, ALK-4, -5, and -7. We hypothesized that this HS-5-induced cell death may be specific to the DNA synthesis phase of the cell cycle. Thus, in order to elucidate how and when TGF- $\beta$  signaling stunts cell growth, we sought to 1) examine levels of potential TGF- $\beta$ -regulated cell cycle proteins using western blotting and 2) measure potency of HS-5-induced death at specific phases of the cell cycle using flow cytometry and live/dead analysis. Our results indicate TGF- $\beta$  downregulates cyclins and stimulates Smad phosphorylation, which correlates with decreased cell growth. Further, HS-5 conditioned media induces the highest levels of toxicity as PCa enters S phase. These findings demonstrate that bone colonization is a dynamic reciprocal interaction between bone stromal and PCa cells mediated, in part, through paracrine signaling by TGF- $\beta$  family members resulting in either rejection or dormancy in the early colonization of the bone microenvironment.

**Jonais RM, Dashner EJ, van Golen KL**

**Abstract**

Signaling through the IGF-1 receptor was studied and it was found that multiple Rho GTPases were activated that were involved in cell survival, motility, and invasion. During a specific study the PC-3 cell line was used to observe the effect of anti IGF-1R treatment on the PC-3 cells. Rho C was inhibited through anti IGF-1R treatment and caused distinct morphological changes as well as changes in the expression level of proteins involved in the invasive capabilities of the metastatic PC-3 cell line. This initiated a study of IGF-1R signaling as well as the expression of Rho C in the LNCaP, C4-2, and the C4-2B (B4) cell lines, which are other metastatic prostate cancer lines that serve as an enhanced model of prostate cancer progression in humans. The results show that the expression of Rho C increases with the level of metastatic capability of prostate cells; the more metastatic cell lines, C4-2 and the C4-2B, have a higher expression of Rho C then the LNCaP cell line. In the B4 cell line, a decrease in the activation Akt is shown when cells are treated with anti IGF-1R treatment. The B4 cell line also showed a decrease in growth when treated with an antibody targeted against the IGF-1R. It is concluded that anti IGF-1R treatment decreases the activation of Akt and that if either Akt or Rho C are inhibited, it may lead to a decrease in metastatic capability of prostate cancer cells. It is also concluded that when the B4 cell line is treated with 3B7, growth is decreased.

# Controlling Prostate Cancer Cell Colony Formation with Next Generation Antiandrogens

Jhoneil Cooper, Gabriella Uceda, John T. Koh;  
Lincoln University, The Department of Chemistry and Biochemistry, The University of Delaware.



## Abstract

Prostate cancer remains the second leading cause of cancer death in men. Anti-androgens are commonly used in the treatment of prostate cancer. Anti-androgen failure can be caused by incomplete AR inactivation by anti-androgens, androgen receptor mutations, androgen receptor over expression and cytokine signaling or cross-talk. The Koh lab has developed a series of compounds in an effort to produce a drug candidate, which will avoid formation of antiandrogen resistant. In this study we perform cell colony formation assays with LNCaP and CWR-22RV1 cells. The analogs PLM1, PLM6 and PAN52 were compared to Bicalutamide (Bic) the active ingredient of Casodex. Pictures were taken using a microscope and the number and size of colonies were compared. At  $0.5\mu\text{M}$  concentration LNCaP cells treated with PLM1 ( $2 \pm 1$ ) showed fewer colonies than PLM6 ( $7 \pm 0.9$ ) and both show fewer than Bic ( $5 \pm 2.4$ ). However PAN52 ( $7 \pm 1.9$ ) when compared to Bic has more colonies. CWR-22RV1 cells treated with PLM1(0) showed fewer reduced colonies than PLM6 ( $0.1 \pm 0.3$ ), both had fewer colonies than Bic( $4 \pm 0.7$ ). However PAN52 ( $1.9 \pm 1.1$ ) had more colonies than Bic but less than the vehicle control ( $2.1 \pm 1.4$ ). Under those conditions Bic showed a decrease in the number of colonies when compared to vehicle control. PLM1 and PLM6 showed a reduced number colonies when compared Bic. PAN52, however had more colonies when compared to Bic but showed a reduced in the number of colonies compared to vehicle. Further studies using higher concentrations of ligand that more closely match ligand IC<sub>50</sub>'s are ongoing.

## Introduction

Prostate cancer remains the second leading cause of cancer deaths in men. Androgen stimulates the growth of androgen-dependent prostate cancer through the activation of androgen receptor (AR) protein. Androgens are hormones (such as testosterone) that are important for normal male sexual development before and during puberty. Androgen receptors allow the body to respond appropriately to these hormones. The receptors are present in many of the body's tissues, where they attach (bind) to androgens. The resulting androgen-receptor complex then binds to DNA and regulates the activity of androgen-responsive genes. By turning the genes on or off as necessary, the androgen receptor helps maintain normal prostate homeostasis. Androgens and androgen receptors also have other important functions in males and females, such as regulating hair growth and sex drive. Androgens are recently refractory cancers. Prostate cancer growth is initially androgen-dependent, and androgen-castration has been the standard treatment for non-metastatic prostate cancer. Advanced Prostate can be treated with antiandrogens. However, the effect of hormonal therapy is temporary, and most tumors become "androgen refractory" (stop responding to androgen-ablation therapy) within a few years. This presents a major obstacle in the treatment of metastatic prostate cancer. Treatment of PCA decades has relied on anti-androgens including testosterone hypothyroidism. However, 30%-40% of patients treated with current antiandrogens hormone inhibitors become resistant between 2-5 years of treatment.

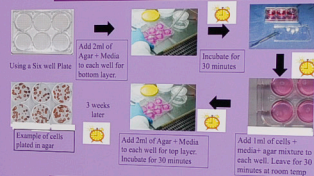
## Antiandrogens



## Objective

To evaluate PLM1, PLM6, PAN52 versus Bicalutamide in soft agar colony formation assays with CWR22 and LNCaP cells and to determine the ability of the cells to grow, form colonies and also their ability to form resistant colonies in the presence of antiandrogens.

## Method



## Data and Results

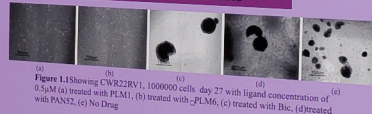


Figure 1.1 Showing CWR22RV1, 100000 cells day 27 with ligand concentration of 0.5µM (a) treated with PLM1, (b) treated with PLM6, (c) treated with Bic, (d) treated with PAN52, (e) No Drug

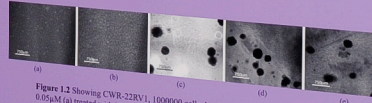


Figure 1.2 Showing CWR-22RV1, 100000 cells day 27 with ligand concentration of 0.05µM (a) treated with PLM1, (b) treated with PLM6, (c) treated with Bic, (d) treated with PAN52, (e) No Drug

CWR-22RV1 cells were cultured in RPMI 1640 with 10% fetal bovine serum (FBS), 2mM L-glutamine and 2mM penicillin streptomycin. The cells were plated in triplicates using concentrations of 25000, 50000 and 100000 cells. Two different concentrations of ligand ( $0.5\mu\text{M}$  and  $0.05\mu\text{M}$ ) were used. Pictures were taken for four weeks using a microscope where the number and size of colonies were counted. During this time the plates seeded with 25000 cells were discarded as the number of cells was too little. CWR22RV1 cells treated with PLM1(0) showed fewer reduced colonies than PLM6 ( $0.1 \pm 0.3$ ), both had fewer colonies than Bic ( $4 \pm 0.7$ ). However PAN52 ( $1.9 \pm 1.1$ ) had more colonies than Bic but less than the vehicle control ( $2.1 \pm 1.4$ ).

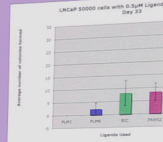


Figure 1.3 Showing LNCaP 50000 cells day 33 with

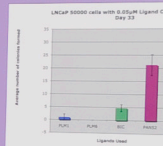


Figure 1.4 Showing LNCaP 50000 cells day 33 with LNCaP cells were cultured in T Medium with 5% 2mM L-glutamine and 50mg/ml Gentamicin. The using concentration of 25000, 50000, and 100000 concentrations of ligand ( $0.5\mu\text{M}$  and  $0.05\mu\text{M}$ ) were used for four weeks using a microscope where the number of colonies were counted. During this time the plates seeded with over grown and therefore that concentration was discarded. The concentration cells treated with PLM1 ( $3 \pm 1$ ) showed fewer colonies than PLM6 ( $7 \pm 0.9$ ) and both show fewer than Bic ( $5 \pm 2.4$ ). However PAN52 ( $7 \pm 1.9$ ) when compared to Bic has more colonies.

## Conclusion

We determined that the compound PAN52 had fewer colonies than no drug at  $0.5\mu\text{M}$  ligand concentration. However PLM1 and PAN52 did not perform as well as the vehicle control. When compared to Bic had fewer colonies. Further studies using ligand concentrations that more closely match IC<sub>50</sub>'s are ongoing.

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<http://www.mckay.edu/covd/Administrative>  
<http://www.syntheticon.com/products/matrix>

## Acknowledgement

We thank Gabriella Uceda for her expertise in the Department of Chemistry and Biochemistry.



# The Effects of Neurotransmitters in regulating Osteocyte Activity: Is There a Bone cell-Neuronal Network?

Jennifer Gray, Mary Buggy\*, and Randall Duncan\*

\*University of Delaware, Newark, DE 19713, \*Department of Biological Sciences, University of Delaware, Newark, DE 19713



NBRE  
Delaware

## Abstract

The effects of neurotransmitters on osteocyte activity were investigated using a novel method of measuring intracellular ATP levels in osteocytes. The results show that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used.

## Introduction

Osteocytes are the most numerous cells in bone and are responsible for the regulation of bone metabolism. They are located within the bone matrix and are surrounded by a network of canaliculi. This network allows for the exchange of nutrients and waste products between the osteocytes and the blood supply. The results of this study suggest that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used.

## Hypothesis

## Materials and Methods

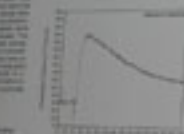


Figure 1: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.

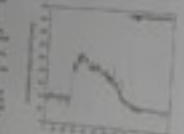


Figure 2: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.



Figure 3: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.

## Results



Figure 4: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.



Figure 5: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.



Figure 6: Effect of ATP release on osteocyte activity. The y-axis represents ATP release (pmol/min) and the x-axis represents time (min). The graph shows a sharp increase in ATP release at time 0, followed by a gradual decline.

## Summary

The results of this study suggest that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used. The results also suggest that the regulation of osteocyte activity is dependent on the type of neurotransmitter used.

## Conclusions

The results of this study suggest that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used. The results also suggest that the regulation of osteocyte activity is dependent on the type of neurotransmitter used.

## Future Work

The results of this study suggest that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used. The results also suggest that the regulation of osteocyte activity is dependent on the type of neurotransmitter used.

## Acknowledgements

The authors thank the following individuals for their assistance in this study: Dr. John Doe, Dr. Jane Smith, and Dr. Bob Johnson.

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Johnson, B., Doe, J., and Smith, J. (2005). The effects of neurotransmitters on osteocyte activity. *Journal of Bone and Mineral Research*, 20(1), 1-10.

## Abstract

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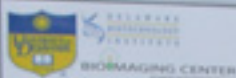
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## Materials and Methods

The effects of neurotransmitters on osteocyte activity were investigated using a novel method of measuring intracellular ATP levels in osteocytes. The results show that neurotransmitters can regulate osteocyte activity and that this regulation is dependent on the type of neurotransmitter used.





# The Role of the microRNA Endonuclease Dicer in Sea Urchin Embryology

Odnaka G. Anyanwu<sup>1</sup>, Deborah M. Powell<sup>2</sup>, Jia L. Song<sup>3</sup> and Kirk A. Coymen<sup>1</sup>

<sup>1</sup>University of Pennsylvania, Gathers, PA  
<sup>2</sup>Delaware Biotechnology Institute, Biomimetic Center, University of Delaware, Newark, DE  
<sup>3</sup>Department of Biological Sciences, University of Delaware, Newark, DE



## Abstract

MicroRNAs (miRNAs) are small non-coding RNA molecules that regulate gene expression. They are involved in many biological processes, including development, differentiation, and disease. In this study, we investigated the role of the microRNA endonuclease Dicer in sea urchin embryology. We found that Dicer is essential for the development of sea urchin embryos, and that its expression is regulated by the transcription factor Pax6. Our results suggest that Dicer plays a critical role in the development of sea urchin embryos, and that its expression is regulated by Pax6.

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## Materials and Methods

**Sea Urchin Embryos.** Sea urchin embryos were collected from the Delaware Biotechnology Institute. The embryos were cultured in seawater and maintained at 15°C. The embryos were collected at various stages of development, including fertilized eggs, cleavage stages, and gastrulae. The embryos were then analyzed by various methods, including RT-PCR, Western blotting, and immunofluorescence.

## Results

**Dicer Expression in Sea Urchin Embryos.** We first investigated the expression of Dicer in sea urchin embryos. We found that Dicer expression was highest in the gastrula stage and decreased in the neurula stage. This suggests that Dicer plays a critical role in the development of sea urchin embryos, and that its expression is regulated by the transcription factor Pax6. Our results suggest that Dicer plays a critical role in the development of sea urchin embryos, and that its expression is regulated by Pax6.

## Results

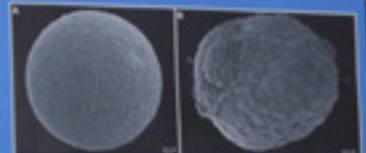


Figure 1. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

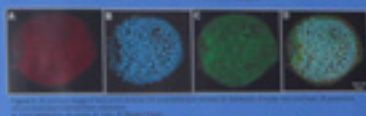


Figure 2. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

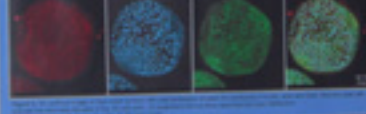


Figure 3. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.



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Figure 5. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

## Results Continued



Figure 6. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.



Figure 7. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.



Figure 8. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

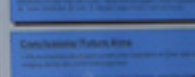


Figure 9. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

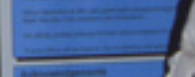


Figure 10. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

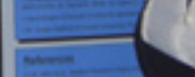


Figure 11. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.



Figure 12. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

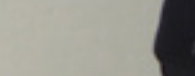


Figure 13. Dicer expression in sea urchin embryos. (a) Fertilized egg, (b) cleavage stage, (c) gastrula stage, (d) neurula stage.

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## UD students excel at UMBC undergraduate research symposium

11:17 a.m., Nov. 8, 2010—Seven University of Delaware students, and one Delaware State University student who is doing research at UD, won top awards at the 13th annual Undergraduate Research Symposium in the Chemical and Biological Sciences at the University of Maryland, Baltimore County, on Oct. 30.

Supported by the National Institutes of Health (NIH), the research conference was devoted entirely to contributions from undergraduates from universities and colleges across the Mid-Atlantic region. Students presented the results of their work in chemistry, biology and at the chemistry-biology interface in poster sessions. All entries were judged in groups of about 5-7 posters with the two top-rated entries in each disciplinary group receiving awards.

Accompanying the UD students was Hal White, professor of chemistry and biochemistry and director of the UD Howard Hughes Medical Institute (HHMI) Undergraduate Science Education Program, which sponsored the students' travel to the event.

"This is the fifth year we have brought students to the UMBC Symposium, and every year the students have done extremely well," White said. "Their success is a real feather in the cap for the Undergraduate Research Program at Delaware. Several of these students will be going on to present their work at the national Experimental Biology Meetings next April in Washington, D.C."

Fourteen UD students and one DSU student participated:

- Erica Boetefuer, "The role of *ATG18* in signal transduction pathways during *Drosophila* development," 2nd place, Biology Group 1L. Adviser Erica Selva.
- Michael Brister, "The structural characteristics of synthetically glycosylated Tau protein sequences," 2nd place, Biochemistry Group 2P. Adviser Neal Zondlo.
- Amy Chevalier, "Trafficking patterns of adenosine A2A receptor," Chemical Engineering. Adviser Anne Robinson.
- Kristofer Dewberry, "Determining the capacity of pulmonary cells to exit the lung during acute influenza virus infection," Animal Science, for work at the University of Pennsylvania School of Medicine. Adviser Gudrun Debes.
- Timothy Gilpatrick, "Examining the binding properties of the enzyme LP-PLA2 and investigating its correlation with coronary heart disease," Biochemistry. Adviser Brian Bahnson.
- Soma Jobbagy, "Characterization of next generation anti-androgens as potential prostate cancer therapeutics," Biochemistry. Advisers Robert Sikes and John Koh.
- Matthew King, "Fibronectin appears in distinctive patterns in the lens of the eye," 1st place, Biology Group 1K. Adviser Melinda Duncan.
- Sanjana Luther, "Comparing the immune response of C57BL6 and BALB/c mice infected with *Vibrio cholera*," Biology. Adviser Michelle Parent.
- Chet Markwalter, "Elucidating mechanisms of heterologous neurokinin 2 receptor expression and trafficking in *S. cerevisiae*," Chemical Engineering. Adviser Anne Robinson.
- Suranjit Mukherjee, "Synthesis of silver nanoparticles for use in an animal model," 2nd place, Biology Group 1J. Adviser Anja Nohe.
- Tejal Naik, "Development of a peptide nucleic acid based siRNA delivery system," 2nd place, Biochemistry Group 2N. Adviser Millicent Sullivan.
- Victoria Roop, "Characterization of the EMT response in CRYBB2PHILmutants," 1st place, Biology Group 1I. Adviser Melinda Duncan.
- Robert Sheehan, "The effects of histone modification on lens fiber cell denucleation," Biology. Adviser Melinda Duncan.
- Navpreet Tung (Delaware State University), "Complex interactions between bone stroma and prostate cancer cells is mediated through TGF-Beta signaling," 1st place, Biology Group 2G. Adviser Robert Sikes.
- Devan Turner, "Vesicle formation through encapsulation of biologically-compatible ionic liquids," 2nd place, Chemistry Group 2B, for work completed as an HHMI Exceptional Research Opportunities Program (EXROP) student with Isiah Warner at Louisiana State University.

The HHMI Undergraduate Science Education Program at UD has several components, one of which is to strengthen undergraduate research in the biomedical sciences. Last summer, the program supported 25 UD students working in faculty laboratories in biology, chemistry, biochemistry, chemical engineering, mathematics and physical therapy.

Earlier this year, UD was one of 50 research universities nationwide to receive a grant from the Howard Hughes Medical Institute (HHMI) for innovative programs to strengthen undergraduate and precollege science education. The four-year grant, which began Sept. 1, is the fifth HHMI award that UD has won.



University of Delaware students excelled at the Undergraduate Research Symposium at the University of Maryland, Baltimore County, Oct. 30. Pictured are, front row, from left, Michael Brister, Sanjana Luther, Amy Chevalier, Erica Boetefuer, Suranjit Mukherjee, Tejal Naik, Robert Sheehan and Devan Turner, and, back row, from left, Navpreet Tung (Delaware State University), Soma Jobbagy, Chet Markwalter, Kristofer Dewberry, Timothy Gilpatrick and Matthew King.

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The Academy Building  
105 East Main Street  
University of Delaware  
Newark, DE 19716 • USA  
Phone: (302) 831-2792  
email: [ud-ocm@udel.edu](mailto:ud-ocm@udel.edu)  
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